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The Scientific Board of the California Medical Association presents the following inventory of items of progress in pediatrics. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist the busy practitioner, student, research worker or scholar to stay abreast of these items of progress in pediatrics which have recently achieved a substantial degree of authoritative acceptance, whether in his own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Pediatrics of the California Medical Association and the summaries were prepared under its direction.

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Cat-Scratch Disease

CAT-SCRATCH DISEASE (CSD) is a disorder characterized by a primary lesion at a site of injury, subacute regional lymphadenitis and mild constitutional symptoms. Complications are rare, the most common being Parinaud oculoglandular syndrome, thrombocytopenia and transient encephalitis. There have been no fatalities in which CSD has been shown conclusively to be the cause. Treatment is generally supportive; antibiotics do not hasten resolution of the disease. In some instances, suppurating fluctuant nodes require aspiration to promote healing. Occasionally, surgical excision of an involved lymph node is required for cure.

Since CSD is benign and self-limited in most cases, it is of interest to clinicians primarily because it is confused with treatable infectious disorders (for example, staphylococci and streptococci infections and tularemia and tuberculosis) which cause regional lymphadenopathy, and other more serious conditions such as lymphomas or sarcoidosis.

CSD should be suspected in a patient with a cutaneous lesion resembling a furuncle and re-

gional lymphadenopathy appearing 1 to 2 weeks after trauma. The diagnosis can be more securely established if (1) there is a history of cat scratch or bite; (2) laboratory studies for other causes of lymphadenopathy are negative; (3) histopathology of a lymph node biopsy specimen is characteristic of CSD, and (4) skin hypersensitivity to cat-scratch antigen can be shown. The presence of three out of four of the above criteria is generally considered sufficient in typical cases. Unfortunately, the etiologic agent of CSD has not been isolated and serological methods of diagnosis are not available. An unequivocal diagnosis of CSD therefore can not be made.

The skin-test antigen is prepared from purulent material aspirated from an affected lymph node. Presumably, it contains the causative agent of CSD, thereby evoking a cutaneous hypersensitivity response in an infected person. Administration and interpretation of the skin test is similar to that used for tuberculin testing.

Recently there has been a growing concern among clinicians that the injection of this human material into man may provide a potential hazard through transmission of slow or incomplete vi-

ruses, or other unknown factors. Indeed, electron microscopic examination of lymph nodes from eight patients with CSD has shown the presence of herpes-like agents in all. The association of viruses from this group with lymphoproliferative disorders in humans and subhuman primates has been recognized for many years. In order to provide a safeguard against possible infection with adventitious agents, it has been suggested that all cat-scratch antigen be exposed to a "sterilizing dose" of gamma radiation (cobalt-60, 2.5 mr) before use. This proposal seems well-founded and reasonable. Although radiation of the antigen reduces the intensity of the cutaneous response in about a third of patients, modification of the criteria for a positive reaction should overcome this problem.

At present, therefore, it is suggested that the diagnosis of CSD can be established in most cases on epidemiological and clinical grounds, together with elimination of other infectious causes through appropriate laboratory methods. This must include examination for the presence of bacteria, both aerobic and anaerobic, fungi, mycobacteria and viruses through smear, culture, serological and skin testing. If this is not sufficient, a biopsy specimen of an involved node should be taken for histological examination. Only under unusual circumstances should it be necessary to carry out a skin test to increase confidence in the diagnosis. Until the safety and reliability of the irradiated skin test material have been established, its use also should be limited.

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The Fetal Hydantoin Syndrome

An association between maternal ingestion of hydantoins during pregnancy and a specific pattern of malformation in offspring was described by Hill and co-workers in 1974 and further delineated by Hanson and co-workers, who referred to this disorder as the fetal hydantoin syndrome. The most frequently noted features of this condition include the following: mild to moderate growth deficiency usually of prenatal onset; mild

mental deficiency; craniofacial defects which include microcephaly, a wide anterior fontanel in the newborn period, ocular hypertelorism, a broad depressed nasal bridge with a short nose, low set abnormally shaped ears and a broad alveolar ridge; limb defects including hypoplasia of the distal phalanges with small nails, finger-like thumbs and altered palmar creases; umbilical and inguinal hernias; short neck with a low hairline, and anomalies of the rib, sternum or spine. Less frequent anomalies include cleft lip or palate, cardiovascular defects, duodenal atresia, anal atresia and genital anomalies.

Hanson and co-workers recently have reported the results of a prospective study of 35 infants whose mothers had been treated with hydantoin anticonvulsants during pregnancy. Eleven percent had the fetal hydantoin syndrome while an additional 31 percent had some features compatible with the prenatal effects of hydantoins, the most frequent of which was developmental or mental deficiency. At present, a safe dose of hydantoin anticonvulsants has not been established below which there is no increased teratogenic risk.

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Dangerous or Ineffective Antidotes

MEDICAL SCIENCE sometimes finds that treatments or procedures that have been used for several years actually are ineffective, perhaps even harmful. Such is the case with three "antidotes" that are widely used and suggested by many toxicology texts, first-aid manuals and product labels: the use of table salt in water as an emetic, using acid solutions to neutralize caustic alkalis and the use of the "universal antidote."

Using table salt in water as an emetic has been suggested for decades. Numerous reports of fatalities caused by this seemingly safe emetic can be found in the literature. The ingestion of a hypertonic saline solution will lead to an elevation in serum sodium concentration, particularly in children. This increase in the effective serum osmolarity will promote a shift of water from the intracellular to extracellular spaces—leading to